West Nile Virus Encephalitis in a Recent Kidney Transplant Recipient: An Unusual Presentation

Dear Editor,

West nile virus (WNV) encephalitis presents diagnostic challenges due to atypical manifestations.[1,2] We report a case of locally acquired WNV encephalitis in a 24-year-old recent kidney transplant recipient.

Six weeks after living-related donor kidney transplantation, the patient presented with abdominal pain and vomiting. Within 48 hours, he developed high-grade fever and rapidly deteriorating sensorium. MRI brain showed T2 FLAIR hyperintensities in the midbrain, pons, thalami, and temporal cortex. Cerebrospinal fluid (CSF) analysis, well documented in transplant recipients[3], revealed pleocytosis (147 cells/µL, 85% neutrophils).

Serology testing showed positive results for both WNV (IgM ELISA, OD: 9.66, Cut off: 0.36) and Japanese encephalitis (JE) virus (IgM ELISA, OD: 1.91, Cut off: 0.45). While JE positivity was potentially due to cross-reactivity with WNV and other flaviviruses, the significantly higher optical density values for WNV suggested it as the primary infection. The importance of confirmatory testing in transplant recipients has been previously highlighted.[4] This was later confirmed by repeat WNV IgM ELISA and plaque reduction neutralization testing at NIV, Pune. Extensive viral panel testing, including for HSV-1, HSV-2, dengue, adenovirus, and influenza A&B, were negative in various samples (serum, CSF, nasopharyngeal swab, and urine). The patient had no travel history to endemic regions but resided in an urban area where WNV cases were reported.

Our case highlights several important points: first, WNV infection in transplant recipients may present atypical initial gastrointestinal rather than neurological symptoms. Second, serological cross-reactivity between flaviviruses can complicate diagnosis. Third, local transmission should be considered even in non-endemic areas.

The patient developed multiple complications, including carbapenem-resistant bacteremia and *C. difficile* colitis. Despite aggressive management, he remained neurologically impaired (Glasgow Coma Scale E2VTM1), requiring prolonged intensive care. The neurological outcomes in WNV infection can be variable and often poor.[5]

This case underscores the importance of considering WNV in transplant recipients presenting with neurological deterioration, regardless of initial symptoms or endemic status of the region.

Conflicts of interest: There are no conflicts of interest.

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